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Evaluation of Serum VASPIN and Lipid Profile in Iraqi Patients with Acute Coronary Syndrome

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Abstract: Acute coronary syndrome (ACS) is an insufficient supply of the myocardium with oxygenated blood in which atherosclerosis is an essential cause of myocardial ischemia. ACS is a leading cause of mortality and morbidity worldwide, so the aims of our study are to evaluate the correlation between serum vaspin and lipid profile, age, gender, body mass index (BMI), and blood pressure in Iraqi patients with ACS; and validate the hypothesis that a low serum vaspin level serves as an indicator of patients with ACS. Our case-control study included a total of 100 individuals; 75 patients with ACS and 25 healthy controls. A total of 75 patients with ACS (50 male, 25 female) as well as 25 patients with STEMI, 25 patients with NSTEMI, and 25 patients with unstable angina (UA) were enrolled in our study. The mean age and BMI of patients were 54.6 years and 29.5 kg/m2, respectively. All were diagnosed by physicians and taken from the Coronary Care Unit at AL-Yarmouk Teaching Hospital (Baghdad). Venous blood used for measuring serum vaspin, total cholesterol, HDL-VLDL and LDL-cholesterol, triglyceride, and BMI was calculated and also BP was measured. Our results showed that serum vaspin levels were significantly lower in patient groups, while total triglyceride and VLDL-cholesterol levels were significantly higher in patients with ACS than controls. A negatively significant correlation was found between serum vaspin with BMI and total cholesterol. We concluded that serum vaspin showed significant changes with the development and progression of ACS and would be valuable in the assessment of patients with ACS. Vaspin may be used as a predictor of ACS.

Keywords: Acute coronary syndrome, Vaspin, Lipid profile, BMI, Iraq.

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INTRODUCTION

Acute coronary syndrome (ACS) remains a major cause of morbidity and mortality worldwide despite significant advances in prevention and treatment. Myocardial infarction [ST-segment elevation myocardial infarction (STEMI, about 30 %), non-STEMI (NSTEMI, 70 %)] and unstable angina (UA) are two conditions that are included under the term ACS [1]. MI occurs when symptoms occur at rest and there is evidence of myocardial necrosis, as demonstrated by an elevation in cardiac troponin or creatine kinase-MB isoenzyme [1,2]. In 50% to 55% of patients in Sub-Saharan Africa, hypertension was the main risk factor for ACS [3]. Vaspin is a member of the adipocytokines belonging to the serpin family, identified in 2005. Vaspin-visceral adipose tissue-derived serine protease inhibitor (also known as SERPINA12 in serpin nomenclature) is linked to the development of inflammation, obesity, and insulin resistance. Obese subjects had a noticeably increased level of vaspin [4]. Human vaspin is a ~45 kDa protein containing 395 amino acids, which belongs to the serine protease inhibitor family [5]. In arterial plaque disorders, elevated serum vaspin levels were thought to be a preventive mechanism against additional endothelial damage, inflammation, and atherosclerosis. Patients with type 2 diabetes or obesity had comparable findings. Elevated serum vaspin levels in arterial plaque disorders were thought to be a defense mechanism against further endothelial damage, inflammation, and atherosclerosis. Obesity or Type 2 diabetes individuals showed similar findings [6]. Higher vaspin levels are associated with a lower gluteofemoral adiposity and a higher risk of type 2 diabetes (T2D), suggesting that vaspin is a viable clinical predictor of T2D [7]. Although the anti-inflammatory properties of omentin and vaspin have been successfully proved, the exact mechanism of their action is not yet entirely understood [8].

After an ACS patient is admitted, their lipid profile needs to be evaluated during the first 24 hours of admission and then on a recurrent basis until they reach a stable, healthy state [9]. Lipid Research Clinics-Coronary Primary Prevention Trial revealed that lowering total and LDL or bad cholesterol levels significantly reduced CAD. More series of clinical trials using statin drugs have provided conclusive evidence that lowering LDL-cholesterol, reduces the rate of myocardial infarction (MI), the need for percutaneous coronary intervention, and the mortality associated with CAD-related causes [10]. Lipid management plays a crucial role in secondary prevention of ACS. However, a large number of patients are still receiving insufficient treatment and do not achieve the LDL- cholesterol reduction levels recommended by guidelines [11].

ACS clinical outcomes are influenced by non-modifiable characteristics such as age and gender. After controlling for many variables, the higher risk for older men persisted, whereas the higher risk for younger women was no longer significant [12].

The ACS spreads throughout the world, including Iraq. Therefore, extensive and in-depth studies must be conducted on patients with ACS in order to find the best methods of prevention, control, and treatment.

MATERIALS AND METHODS

This case-control study included 100 individuals: 75 patients and 25 control healthy; the criteria of inclusion were: un stable angina pectoris and STEMI and NSTEMI patients with recent acute myocardial infarction admitted into the coronary care unit (CCU) from February to July 2024 at AL-Yarmouk Teaching Hospital (Baghdad-Iraq).

A total of 75 patients (50 male, 25 female) consisting of: 25 patients with unstable angina pectoris (12 male, 13 female), 25 patients with STEMI (22 male, 3 female), and 25 patients with NSTEMI (16 male and 9 female), were enrolled in this study. The consultant clinically examined each patient and electrocardiograph ECG, cardiac enzymes, and cardiac troponin to achieve the diagnosis.

Several modalities of therapeutic agents--anti-angina, anticoagulants, and lipid lowering agents are the cornerstones in the management of ACS patients.

The criteria of exclusion were patients with valvular heart disease, malignant disease, and infectious diseases, inflammatory diseases such as collagen disease, neoplasm, hematological disorders, advanced renal disease, liver disease, and diabetes mellitus.

The control group consisted of 25 healthy persons (14 males and 11 females) participants matched by age and BMI. Clinical examination of patients and controls was done for BP, weight (Kg), and height (m). The BMI was calculated using Quetelet's equation: BMI (kg/m^2) = weight/$(height)^2$ taking the cutoff ≥ 25 kg/m^2 as an indication of overweight-obese.
Ten milliliters (10 ml) of blood were obtained by venipuncture after 10-12 hours of overnight fasting, using a 10 ml disposable syringe between 9.00 and 11.00 a.m. Then centrifuged at 3000 rpm for 10 minutes to collect serum. Serum was divided into aliquots (250 µl) in Eppendorff tubes and stored in a freezer (-20 ºC) until use.

Serum Vaspin Detection Range

The standard curve concentrations utilized for the ELISA's were 200 pg/ml, 100 pg/ml, 50 pg/ml, 25 pg/ml, 12.5 pg/ml, 6.25 pg/ml, and 3.12 pg/ml.

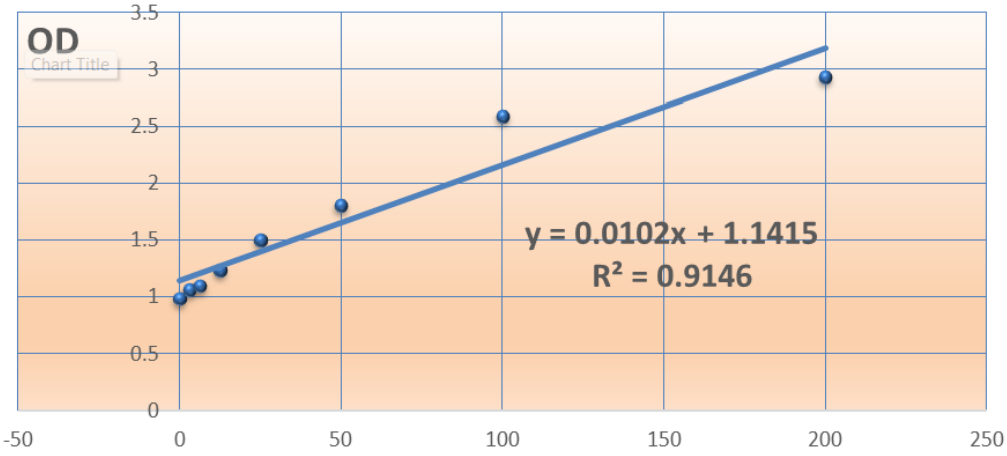

Figure 1: Vaspin concentration (pg/ml).

Assessment of Lipid Profile

Total Serum Cholesterol (enzymatic colorimetric assay)
Serum Triglyceride: (enzymatic colorimetric assay)
High Density Lipoprotein (HDL) Cholesterol (colorimetric method)
Low Density Lipoprotein (LDL) cholesterol and Very Low Density Lipoprotein cholesterol (VLDL): We were determined by using Friedewald's formula.
VLDL-C calculated in mg/dl = TG/5
Calculated LDL-C in mg/dl = Total Cholesterol – (HDL + VLDL)

Statistical Analysis

Analysis of data was carried out using the available statistical package of SPSS-22 (Statistical Packages for Social Sciences, version 22). Data were presented in simple measures of frequency, percentage, mean, standard deviation, and range (minimum-maximum values). The significance of the difference of different means (quantitative data) was tested using the student's t-test for the difference between two independent means, the paired t-test for the difference of paired observations (or two dependent means), or the ANOVA test for the difference between more than two independent means. The significance of differences of different percentages (qualitative data) was tested using the Pearson Chi-square test (χ^2-test) with the application of Yate's correction or the Fisher Exact test whenever applicable.

RESULTS

The mean age and BMI of the 75 participating patients that were recorded were 54.6 years and 29.5 kg/m2, respectively. Of them, 66.6% were male, 29.3% had a family history of acute coronary syndrome, and 42.6% were smokers.

The age and gender of our study participants (in number and percentage) are presented in Table 1, which shows the mean age and gender of seventy five (75) patients divided into three groups: 25 patients with ST segment elevation myocardial infarction (STEMI) with a mean age of 55.1±10.3 (range 39-70) years and consisted of 22 males and 3 females; 25 non ST segment elevation myocardial infarction (NSTEMI) with a mean age of 58.0±9.5 (range 36-70) years and consisted of 16 males and 9 females, 25 Unstable Angina (UA) with a mean age of 55.88±11.2 (range 27-70) years and consisted of 12 males and 13 females .This study included also 25 healthy control subjects with a mean age of 51.2±8.3 (range 37-65) years, and consisted of 14 males and 11 females. The mean age of control was not significantly different from that of the STEMI, NSTEMI, and UA.

Table-1: Age and gender of the study groups.

		STEMI		Non-STEMI		Unstable Angina		Control		P value
		No	%	No	%	No	%	No	%	
Age (years)	<40	1	4.0	1	4.0	2	8.0	2	8.0	0.364
	40---49	9	36.0	4	16.0	4	16.0	9	36.0	
	50---59	4	16.0	8	32.0	9	36.0	9	36.0	
	=>60years	11	44.0	12	48.0	10	40.0	5	20.0	
	Mean ±SD (Range)	55.1±10.3 (39-70)		58.0±9.5 (36-70)		55.8±11.2 (27-70)		51.2±8.3 (37-65)		
Gender	Male	22	88.0	16	64.0	12	48.0	14	56.0	0.021*
	Female	3	12.0	9	36.0	13	52.0	11	44.0	
*Significant difference between proportions using Pearson Chi-square test at 0.05 level.										

The clinical examination of body mass index (BMI) and blood pressure (BP) of the participants is shown in Table 2, which revealed no significant difference between STEMI, NSTEMI, and UA patients and the control group; (28.2±5.6 kg/m^2) (range 19.0-42.6) (28.8±3.8 kg/m^2) (range 20.334.2),(31.5±7.0 kg/m^2) (range 20.8-46.0) and (27.3±4.6 kg/m^2) (range 20.5-37.5) respectively. Regarding blood pressure measurements (systolic and diastolic BP), there were also no significant differences between the same groups. The patients were treated with several modalities of therapeutic agents: anti-angina, anticoagulants, and lipid-lowering agents were the cornerstones in the management of ACS patients.

Table-2: Clinical examination (BMI and BP) of the study groups

	STEMI		Non-STEMI		Unstable Angina		Control		P value
	No	%	No	%	No	%	No	%	
Normal (18.5-24.9)	7	28.0	4	16.0	5	20.0	7	28.0	0.052
Overweight (25-29.9)	7	28.0	9	36.0	5	20.0	14	56.0	
Obese (=>30)	11	44.0	12	48.0	15	60.0	4	16.0	
BMI(Kg/m2) Mean ±SD (Range)	28.2±5.6 (19.0-42.6)		28.8±3.8 (20.3-34.2)		31.5±7.0 (20.8-46.0)		27.3±4.6 (20.5-37.5)		
Weight (Kg) Mean ±SD (Range)	77.9±18.9 (50.0-132.0		78.9±17.3 (56.0-121.0		80.8±15.7 (58.0-112.		77.8±13.3 (53.0-110.0)		
Height (cm) Mean ±SD (Range)	165.6±7.3 (150-180)		164.7±10.5 (147-188)		160.9±8.9 (148-178)		168.9±9.8 (148-182)		
SBP (mmHg) Mean ±SD (Range)	127.5±24.7 (80-180)		127.6±25.1 (95-200)		122.4±25.4 (80-180)		118.6±7.0 (110-130)		0.410
DBP (mmHg) Mean ±SD(Range)	77.7±12.3 (60-110)		74.4±11.6 (55-100)		72.9±16.7 (50-115)		77.0±5.2 (70-90)		0.473

*Significant difference between proportions using Pearson Chi-square test at 0.05 level.

The results of the lipid profile (total cholesterol, triglycerides, HDL-, VLDL-, and LDL-cholesterol) in patients with ACS compared with controls are presented in Table 3.

There are high significant differences between STEMI, NSTEMI, UA patients, and control regarding the serum triglycerides and VLDL-cholesterol. The results of triglycerides (mg/dl) are: STEMI (143.6±58.9), NSTEM (168.7±52.0), UA (129.7±52.1), and control (99.5±29.7). The results of VLDL-cholesterol (mg/dl) are: STEMI (28.7±11.8), NSTEM (33.7±10.4), UA (25.9±10.4), and control (20.0±6.0).

The higher level of serum total cholesterol and LDL-cholesterol in the myocardial infarction (STEMI and NSTEMI) patients compared with control. The results of total cholesterol (mg/dl) are: STEMI (181.9±45.9), NSTEM (185.2±38.6), UA (161.8±28.5), and control (161.3±19.9). The results of LDL-cholesterol (mg/dl) are: STEMI (112.6±43.8), NSTEM (115.0±35.5), UA (94.0±29.4), and control (94.4±20.0). All the ACS patient results were taken at the time of admission to the hospital.

Table-3: Serum lipid profile components in patient with ACS compared to controls

	STEMI	Non-STEMI	Unstable Angina	Control	p-value
Total cholesterol (mg/dl)	181.9±45.9 (115-306)	185.2±38.6 (110-258)	161.8±28.5 (111-228)	161.3±19.9 (123-196)	0.021#
Triglycerides (mg/dl)	143.6±58.9 (56.0-326.0)	168.7±52.0 (79.0-303.0)	129.7±52.1 (49.0-244.0)	99.5±29.7 (51-152.0)	0.0001#
HDL-C (mg/dl)	40.5±11.3 (24.0-61.0)	36.4±8.7 (24.0-53.0)	41.8±13.5 (24.0-76.0)	47.0±13.5 (30.0-72.0)	0.021#
VLDL-C (mg/dl)	28.7±11.8 (11.2-65.2)	33.7±10.4 (15.8-60.6)	25.9±10.4 (9.8-48.8)	20.0±6.0 (10.2-30.4)	0.0001#
LDL-C (mg/dl)	112.6±43.8 (42.4-205.8)	115.0±35.5 (53.6-186.6)	94.0±29.4 (37.6-154.2)	94.4±20.0 (65.0-130.8	0.039#

#Significant difference using ANOVA test at 0.05 level

The results of serum vaspin in patients with ACS compared with controls are presented in Table 4. There are significant differences between STEMI,NSTEMI, UA patients, and control regarding the serum vaspin, the higher level of serum vaspin in the control mean ±SD (97.48±50.16) and reduced in ACS patients at the time of admission to the hospital; the lower level about 14 times in the unstable angina mean± SD (6.82±1.65),

and lower level about 27 times in NSTEMI mean ±SD(3.5±0.73), and the lower level about 38 times in STEMI mean ±SD (2.56±0.65), and show a description of the sample in 5th ,25th,50th,75th,95th,99th.

Table-4: Results of serum Vaspin in patients with ACS compared with controls.

Serum Vaspin (pg/ml)		STEMI	Non-STEMI	Unstable Angina	Control
	Number	25	25	25	25
	Mean ±SD	2.56±0.65	3.50±0.73	6.82±1.65	97.48±50.16
	Standard Error of Mean	0.130	0.146	0.329	10.032
	Range	(0.9-3.2)	(2.6-4.8)	(4.5-9.3)	(17.1-187)
	Percentile 05th	1.4	2.7	4.7	33.0
	25th	2.5	2.9	5.7	61.0
	50th (Median)	2.7	3.3	6.1	85.0
	75th	3.1	4.1	8.5	138.0
	95th	3.2	4.8	9.3	181.0
	99th	3.2	4.8	9.3	187.0
P value compare to Control		0.0001*	0.0001*	0.0001*	-
P value compared to Unstable Angina		0.0001*	0.0001*	-	-
P value compared to Non-STEMI		0.0001*	-	-	-
P value comparing all		0.0001#	-	-	-
*Significant difference between two independent means using student's t-test at 0.05 level					
#Significant difference between three independent means using ANOVA test at 0.05 level					

Statistical analysis of vaspin levels in patients with ACS compared with all markers regarding controls was done using the ANOVA test (between three independent means of one marker) and student's t-test (between two independent means of one marker) using the student's t-test at the 0.05 level and is presented in Table 5.

Table-5: P value of serum vaspin, means using the student's t-test or ANOVA test at 0.05 levels

Vaspin pg/ml	STEMI	Non-STEMI	U Angina	Control
Age	0.836	0.737	0.436	0.390
Gender	0.239	0.470	0.020*	0.670
BMI	0.014*	0.003*	0.0001*	0.051
Family history	0.736	0.114	0.746	-
Smoking	0.567	0.419	0.515	0.182
Cholesterol	0.337	0.505	0.209	-
TG	0.968	0.232	0.786	0.953
HDL-C	0.966	0.358	0.429	0.373
*Significant difference between two independent means using student's t-test or ANOVA test for difference among three independent means at 0.05 level				

DISCUSSIONS

Coronary artery disease is a condition that affects many people and has a high mortality rate. The most frequent cause of myocardial infarction is atherosclerosis, a progressive inflammatory condition of the arterial wall that obstructs the coronary artery or arteries, leading to acute coronary syndromes (ACS), which include STEMI, NSTEMI, and UA [13]. Multiple factors, such as smoking, obesity, hypertension, dyslipidemia, and family history of coronary artery disease, play a crucial role in the pathogenesis of CAD [14]. The mean age of our participant patients (50 male, 25 female) with ACS is 54.6 years. An ever-growing percentage of ACS patients are older individuals [15]. Individuals over 50 are more likely to get acute coronary syndrome (ACS); younger patients may also be impacted. The mortality rate for women is higher than that of males [16].

Obesity is an independent modifiable risk factor for coronary heart disease (CHD) in both genders. Body mass index (BMI) is considered a clinical tool for evaluating obesity. The mean BMI of the patients with ACS was 29.5 kg/m2. Results of this study revealed that coronary artery disease had a significant positive correlation with the BMI. This result agreed with the study of Ratwatte S *et al*. [17], who found that the BMI and the earlier age of the first ACS showed a significant adverse linear connection. When comparing patients with severe obesity to those who were normal weight, the proportion of patients without risk factors was noticeably higher. While Mornar Jelavic M *et al*. [18]

found that obesity is correlated negatively with primary and secondary outcomes and positively with traditional cardiovascular risk variables, confirming the continuation of the "obesity paradox" between the same groups; probably the patients were treated with several modalities of therapeutic agents: anti-angina, anticoagulants, and lipid lowering agents, Our results are consistent with the results of Shlomai G *et al*. [19] and Winzap PA *et al*. [20].

Components of the lipid profile play an important role in cardiovascular risk assessment.

Current data revealed that there was an elevation in serum total cholesterol concentration and serum LDL-C concentration in myocardial infarction patients (STEMI and NSTEMI) as compared to the control group. These results are supported by other studies, which assumed that elevated levels of total and low-density lipoprotein cholesterol (TC, LDL-C) were considered important risk factors for CAD [21-23]. On the other hand, there are other studies with different results, as follows: Following an acute myocardial infarction, there are cyclical variations in the serum lipid profile. The trend that follows is a rise in TGs and a decrease in TC, LDL-C, and HDL-C [9]. The long-term prognosis for patients with ACS was significantly worse for those with low LDL-C levels at admission than for those with high LDL-C values [24]. Our results revealed high significant differences between ACA patients (STEMI, NSTEMI, UA), and control regarding the serum triglycerides and VLDL-cholesterol. This is consistent with the studies of Kumar N *et al*., [9] and Sato R *et al*., [24].

Vaspin's antioxidant and anti-inflammatory functions in the smooth muscle cells of peripheral blood vessels were shown to inhibit the rise in systolic BP [25]. Additionally, vaspin was shown to upregulate the PI3-kinase/Akt signaling pathway, shielding vascular endothelial cells against free fatty acid-induced death [26].

In this investigation, in contrast to the control group, patients with ACS (STEMI, NSTEMI, UA) had significantly lower serum vaspin levels. A negatively significant correlation was found between serum vaspin and BMI, total cholesterol. Serum Vaspin levels were lower in ACS patients in comparison with the control group; serum vaspin showed significant changes with the development and progression of ACS and would be valuable in the assessment of patients with ACS. These results are in agreement with Kobat *et al*., [27]; Zhou *et al*. [28].

More research in a variety of settings and cautious interpretation are needed because of the significant diversity and paucity of studies.

CONCLUSIONS

Elevated serum lipid profile levels in Iraqi ACS patients are considered significant risk factors for coronary artery disease (CAD). Serum vaspin level is significantly decreased in patients with ACS. These findings may help in the diagnosis and treatment of patients at high risk for CAD. Vaspin has the potential to predict ACS. So, following an ACS, a repeat measurement of vaspin should be carried out.

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